Oxygen Extraction Ratio

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OVERVIEW

- Oxygen extraction ratio (O2ER) is the ratio of oxygen consumption (VO2) to oxygen delivery (DO2)
  - global oxygen delivery (DO2) is the total amount of oxygen delivered to the tissues per minute, irrespective of the distribution of blood flow
  - oxygen consumption (VO2) is the total amount of oxygen removed from the blood due to tissue oxidative metabolism per minute
- Under resting conditions with normal distribution of cardiac output, DO2 is more than adequate to meet VO2 and ensure that aerobic metabolism is maintained
- Oxygen that is not extracted returns to the mixed venous circulation
- ScvO2 of 70% indicates oxygen delivery is adequate (assuming normal microcirculatory function)

CALCULATION

O2ER = VO2 / DO2 = (SaO2-SvO2) / SaO2

NORMAL VALUES

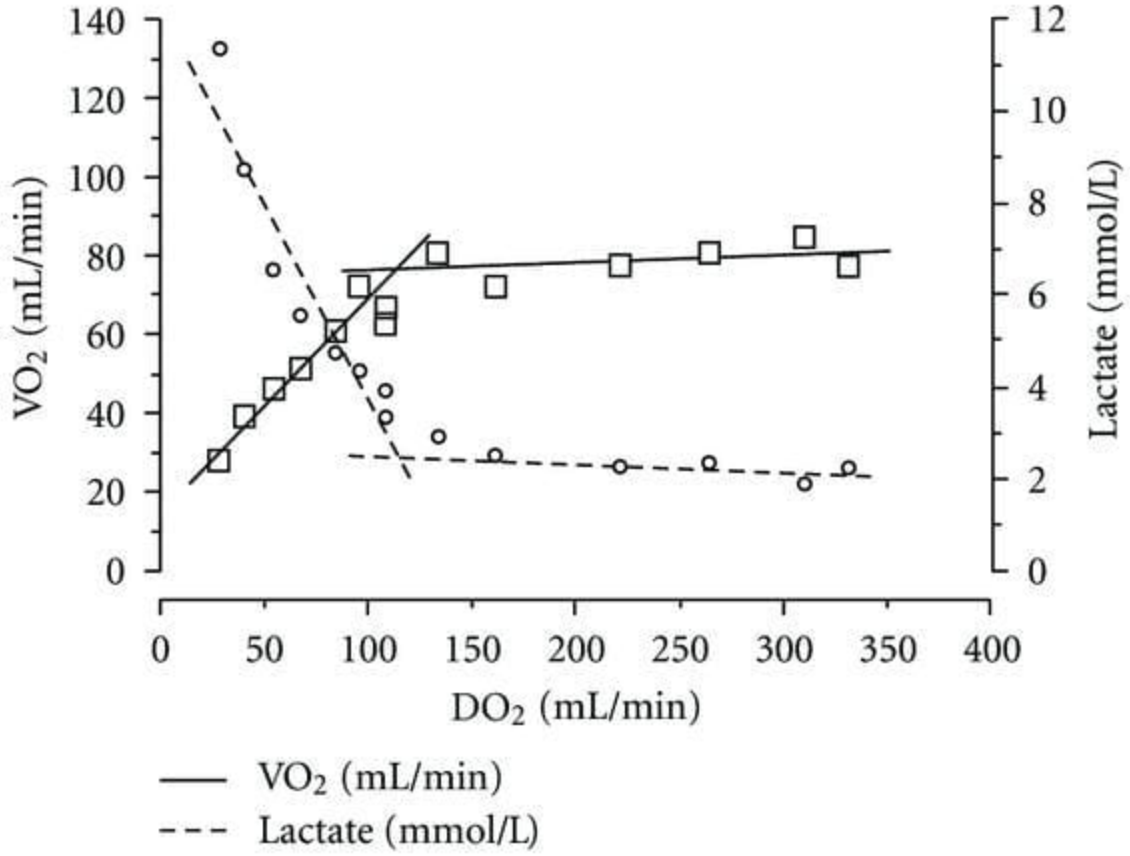
In a normal 75 kg adult undertaking routine activities:

- VO2 is approximately 250 ml/min (cf. VO2max in a non-athlete 75kg person is about 3L/min)
- DO2 is approx 1 L/min
- O2ER is 25% (increases to ~70% during maximal exercise in an athlete)
- SvO2 70%

O2ER varies for different organs:

- cardiac O2ER = >60%
- hepatic O2ER = 45-55%
- renal O2ER = <15%

RELATIONSHIP BETWEEN VO2 and DO2



From Nebout and Pirrachio, 2012

- Initially, as metabolic demand (VO2) increases, or DO2 diminishes, O2ER rises to maintain aerobic metabolism and consumption remains independent of delivery.
- However, at a point called critical DO2 (cDO2)—the maximum O2ER is reached. This is believed to be ~70%.
- Beyond cDO2 any further increase in VO2, or decline in DO2, must lead to tissue hypoxia and anaerobic metabolism (lactate production is a surrogate for this)

In reality each tissue / organ has its own cDO2 — the higher the O2ER for a given tissue , the greater the dependence on DO2 (supply dependence).

INTERPRETATION OF ABNORMAL O2ER

High O2ER suggests inadequate oxygen delivery (OH CRAP; shock)

- oxygen (hypoxic hypoxia: low FiO2 gas or high altitude; lung disease)
- hemoglobin (anemia)
- contractility
- rate/ rhythm
- afterload
- preload
- shock/ hypoperfusion due to other causes

or increased oxygen consumption (VO2)

- fever and inflammatory states, e.g. sepsis, burns, trauma, surgery
- increased metabolic rate, e.g. hyperthyroidism, adrenergic drugs, hyperthermia, burns
- increased muscular activity, e.g. exercise, shivering, seizures, agitation/anxiety/pain, weaning from ventilation/ increased respiratory effort

Low O2ER suggests increased oxygen delivery

- hyperoxia, e.g high FiO2 gas, hyperbaric oxygen or ECMO

or decreased oxygen consumption

- decreased metabolic rate, e.g. hypothyroidism, sedatives/ hypnotics, hypothermia
- decreased muscular activity e.g. sedation/analgesics, muscle paralysis, ventilatory support
- antipyretics
- Starvation/hyponutrition
- Sepsis due to shunting and histotoxic hypoxia
- Histotoxic hypoxia, e.g. cyanide poisoning

References and links

Journal articles

- McLellan SA, Walsh TS. Oxygen delivery and haemoglobin. Contin Educ Anaesth Crit Care Pain (2004) 4 (4): 123-126. [Free Full Text]
- Nebout S, Pirracchio R. Should We Monitor ScVO2 in Critically Ill Patients? Cardiol Res Pract. 2012;2012:370697. PMC3177360.

Critical Care

Compendium

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Chris is an Intensivist and ECMO specialist at The Alfred ICU, where he is Deputy Director (Education). He is a Clinical Adjunct Associate Professor at Monash University, the Lead for the Clinician Educator Incubator programme, and a CICM First Part Examiner.

He is an internationally recognised Clinician Educator with a passion for helping clinicians learn and for improving the clinical performance of individuals and collectives. He was one of the founders of the FOAM movement (Free Open-Access Medical education) has been recognised for his contributions to education with awards from ANZICS, ANZAHPE, and ACEM.

His one great achievement is being the father of three amazing children.

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